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Can HIV alter the quantity-quality switch and delay the fertility transition in Sub-Saharan Africa?

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Abstract According to the conventional theory of the demographic transition, mortality decline has represented the major trigger for fertility decline and eventually sustained economic development. In Sub-Saharan Africa (SSA), the HIV/AIDS epidemic has had a devastating impact on mortality, by dramatically reversing, in high HIV-prevalence countries, the long-term positive trend in life expectancies. Despite the fact that SSA as a whole is suffering a delayed and slow fertility transition compared to other world's regions, and despite evidence for halting or even reverting fertility decline in countries with severe HIV epidemics, there seems to be little concern amongst international policy makers, about the ultimate impact that HIV might have on SSA fertility. This work reports model-based evidence of the potential for a HIV-triggered reversal of fertility in high HIV-prevalent SSA countries induced by the fall in education and human capital investments following the drop in life expectancy for young adults. This eventually breaks down the virtuous circle promoting the switch quantity-to-quality of children. This result suggests that the current evidence on fertility halting and declining education in high HIV-prevalent SSA countries should be seriously taken into consideration to prioritise current international interventions.

Keywords Sub-Saharan Africa, fertility transition, quantity-quality switch, HIV/AIDS epidemics, human capital accumulation, fertility reversal.

JEL Classification J11, J13, O1, O41

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1. Introduction

The current magnitude and shape of human populations worldwide are the outcome of that major process of mortality and fertility decline known as the *demographic transition* (DT). The DT debuted in Europe after 1750 [Chesnais (1987); Livi-Bacci (2017)] by a phase of mortality decline which interrupted the long-term stagnation of western populations around their *Malthusian* regime, characterised by high levels of mortality and fertility. Mortality decline, which was responsible for rapid population growth, was later followed (after 1850) by fertility decline, landing (around 1930) on a *modern* stationary regime at low levels of fertility and mortality [Chesnais (1987); Bulatao and Casterline (2001); Livi-Bacci (2017)]. The other world's regions followed later along their own paths [Bulatao and Casterline (2001); Bongaarts and Casterline (2012)]. Modern demo-economic conceptualizations of the DT emphasise its interplay with the industrial revolution, and the related endogenous nature of mortality decline. The latter triggered fertility decline by promoting investments in education, thus favouring the switch in children's demand from "quantity" (typical of Malthusian regime) to "quality" (typical of the modern regime) [Galor (2011) and references therein] and eventually becoming a main engine of economic development.

In Sub-Saharan Africa (SSA), the less-developed world region also suffering the highest burden from infectious diseases [Bloom and Canning (2004); IHME (2013)], mortality decline was halted in 1980s by the HIV/AIDS epidemic (Fig. 1a). In high-prevalence settings, HIV was able to reverse [UN (2015)] the long-term positive trend of life expectancies (Fig. 1b). The memory of HIV on mortality might persist for long time (Fig. 1b). For example, in Lesotho, the life expectancy at age 15 (e_{15}), which was estimated to fall from 53 to 35 yr during 1990-2010, is projected by the UN medium variant to return to its pre-HIV level only by 2060 (Fig. 1b).

In addition, the fertility transition in SSA, already delayed and slower compared to other world regions departed from similar initial conditions – such as Asia and the Latin America [Bongaarts and Casterline (2012)], with several countries still showing total fertility rates (TFR) about six [UN (2015)] –, is recently experiencing further symptoms of stalling, or even relapsing, fertility [Bongaarts and Casterline (2012); UN (2015)].

Worryingly, most of the countries showing the clearest symptoms of stall in their fertility trajectory are amongst the highest HIV-prevalent countries (Fig. 1c), which notably departed at the onset of the epidemics (1980) from higher income and lower fertility conditions. Evidence of stalling or relapsing fertility at still high TFR levels is clear for Botswana, Zimbabwe, Lesotho and Namibia. On the one hand, in South Africa the TFR, which was fast declining long before 1990, sharply slowed down thereafter. On the other hand, in countries currently suffering intermediate HIV epidemics – most of which were characterised by higher fertility at the onset of the epidemics – fertility decline slowed down during 1995-2005 (e.g., Tanzania, Malawi, Zambia, Mozambique and Kenya) but accelerated thereafter.

Despite this, the dramatic possibility that the injury caused by massive HIV epidemics might compromise major societal processes, such as the fertility decline, is acknowledged neither by observers who pointed out the anomalous fertility decline in SSA [Bongaarts and Casterline (2012)], nor by the last UN population projections [UN (2015)]. Notably, while UN projections include AIDS mortality, possible feedbacks on fertility were deliberately ruled out: "The fertility projections for sub-Saharan Africa follow the general path from high to low fertility observed in other regions." [UN (2015), p. 19]. However, the latter scenario is not dynamically plausible as is shown e.g., by the case of Zimbabwe (Fig. 1d), which seems to postulate a Deus ex Machina suddenly restoring the right fertility pathway at 2020 while the HIV epidemic is still fully on.

This work aims at bringing macroeconomic dynamic model-based evidence of the potential for a HIV-triggered reversal of fertility in high HIV-prevalent SSA countries induced by the fall in education and human capital investments following, in turn, the drop in life expectancy for young adult individuals. Our results show, in particular, that the potential for a paralysis in the fertility

transition is a robust phenomenon occurring even under rather optimistic hypotheses on the effects of control programs currently in progress in SSA.

The rest of the article proceeds as follows. Section 2 describes the (macro)economic background underlining the interplay between HIV/AIDS and fertility. Section 3 builds on the macro-dynamic model and discusses the parametrisation used in the numerical simulations. Section 4 reports the main results. Section 5 outlines the conclusions.

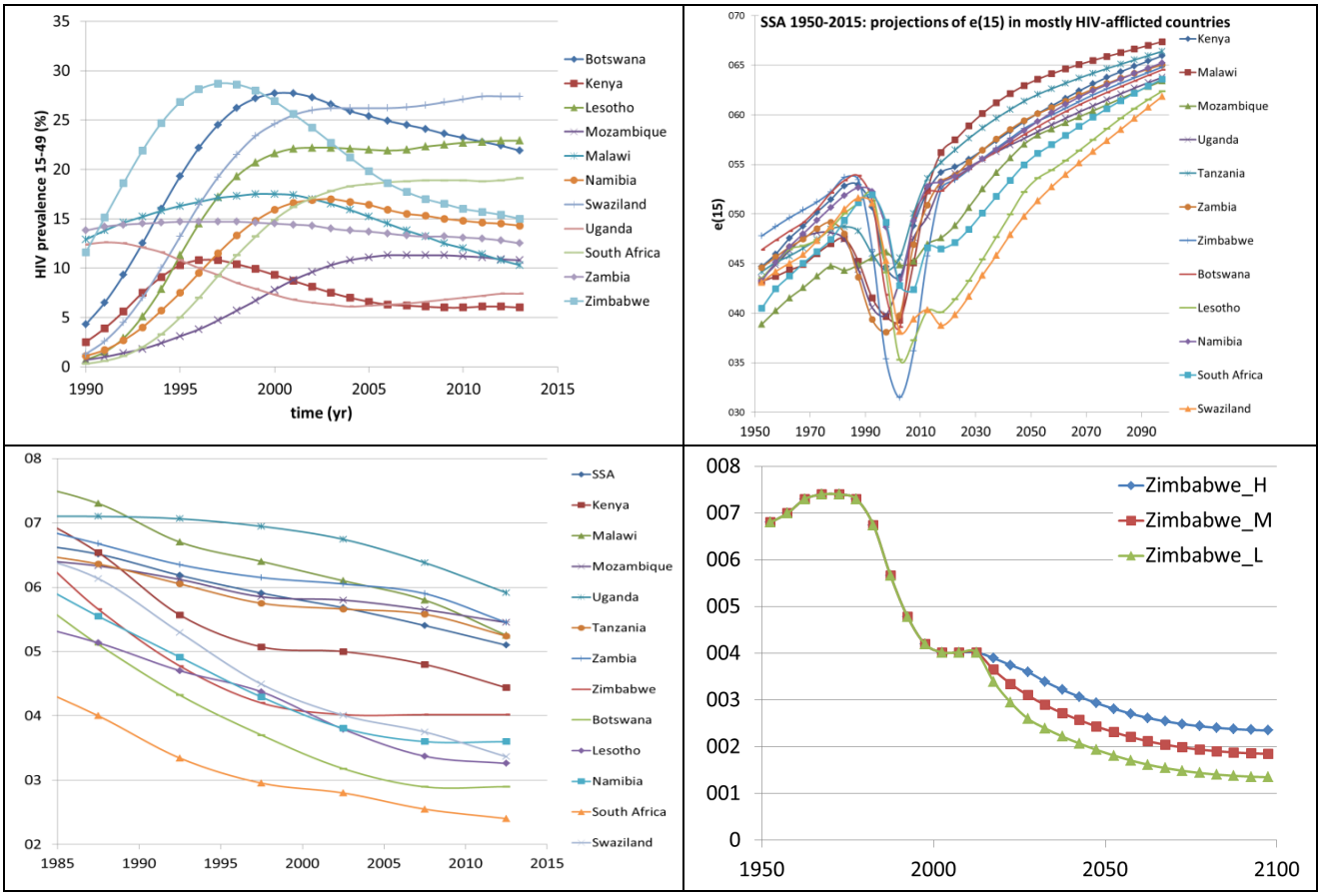

Fig. 1. Demographic trends in SSA countries with higher HIV prevalence: (a) Estimated trend of HIV prevalence in 15-49 yr old individuals [source: (UNAIDS) 2016]; (b) estimated and projected future evolution of mortality of young adults, as measured by e_{15}, the life expectancy at 15 years [source: UN (2015)]; (c) estimated trend 1980-2015 in the total fertility rate (TFR), [source: UN (2015)]; (d) the case of Zimbabwe: projected future evolution of TFR according to UN projection variants (M="medium", L="low", and H="high") [source: UN (2015)].

2. The economic literature on the impact of HIV/AIDS of fertility and development in SSA

Given the critical role of fertility decline as an engine of development, a major conundrum regards the ultimate impact of HIV on the pace and extent of the fertility transition. Two major opposite scenarios have then emerged on this issue in the economic literature. At the one extreme, [Young (2005)] reaches the conclusion that AIDS, once brought under control, will allow future generations to enjoy higher welfare than current ones. This scenario, remindful of the "world of opportunities after the Black death", which allowed an epoch of sustained growth in 15th century Europe, would eventually prevail because of the increase in the capital- and output-labour ratios due to the direct and indirect effects of AIDS mortality on labour supply. This conclusion, based on a model à la Becker with children quantity-quality trade-off and fixed saving rate as in Solow (1956), holds both in the medium term, due to the mortality of young individuals, and in the longer term, due to the fertility decline allowed by the increased female labour market participation [Young (2005, 2007)].

These effects will dominate the main negative one of HIV i.e., disruption of human capital [Young (2005)].

At the other extreme, [Kalemli-Ozcan (2012)] shows a positive (resp. negative) correlation between HIV prevalence and fertility (resp. school enrolment) and then highlights the risk that HIV might reverse the fertility transition in SSA. This is because of the upward pressure that the upturn in mortality due to AIDS has on the precautionary demand for children (i.e., the preference for "quantity"), and to the downward pressure on the demand for their education. This scenario was further supported in [Juhn et al. (2013)]. Given these two opposite scenarios, where fertility will actually land is a major question.

In between these two polar positions, there is a number of further theoretical demo-economic contributions [Corrigan et al. (2005); Boucekkine et al. (2009); Boucekkine and Laffargue (2010); Chakraborty et al. (2010, 2016); Boucekkine (2012); Bell and Gersbach (2013); Azomahou et al. (2016)]. Corrigan et al. (2005) study an OLG growth model where the large number of orphans caused by the HIV/AIDS epidemics negatively affects accumulation of both human and physical capital. Boucekkine et al. (2009), based on a theoretical OLG economy where HIV/AIDS acts as an exogenous shock, show by an empirical analysis that the epidemic unambiguously affects net fertility in SSA. Nevertheless, the only authors in the literature that explicitly account for a dynamics of HIV/AIDS infection in a macroeconomic set up are [Chakraborty et al. (2010, 2016)]. Their works are important as they innovate on the side of the interplay between disease variables and economic variables, but they avoid to incorporate a mechanism that endogenously make HIV/AIDS responsible for affecting population variables. In other words, and more generally, none of the above mentioned works consider an economy where an infectious disease with high long-term morbidity, such as HIV/AIDS, affects individual decisions with regard to fertility (directly) and mortality (indirectly through education investments). This work aims at contributing to this debate by a novel model integrating, for the first time in the literature, a simplified representation of HIV spread through sexual transmission [Chakraborty et al. (2010, 2016)] into a Unified Growth Theory overlapping generations (OLG) framework [Galor (2011); Galor and Weil (1996, 2000)], including fertility and education decisions under endogenous child and adult mortality. The model corroborates the empirical results on fertility reversal due to HIV/AIDS obtained by [Kalemli-Ozcan (2012)].

3. The macroeconomic dynamic framework

The key ingredient of our model is represented by the assumption of endogenous child and adult survival probabilities in the absence of HIV. These are taken as appropriately parametrised increasing functions of human capital accumulation in order to reproduce, in the absence of HIV, a regular demographic transition pattern triggered by the interplay between increasing survival and the level of income and investments in education, aiming to reflect the pre-AIDS setting in SSA countries, where the demographic transition was ongoing prior to HIV onset. In the presence of HIV these survival probabilities are assumed to scale with infection prevalence to mirror the disruption of human capital caused by AIDS mortality. Unlike previous economic works on the demographic transition [Galor (2011); Galor and Weil (1996, 2000)], principally stressing the importance of child mortality, the critical factor of the model responses is represented by the effect of HIV on adult mortality, whose upsurge reduces the resources for education [Kalemli-Ozcan (2012)], eventually eroding the growth of human capital.

3.1. The model

We consider an OLG closed economy accounting for fertility, mortality, education and human capital accumulation along the line of the economic literature of the Demographic Transition [Galor (2011)]. The economy is populated by a continuum of rational and identical individuals of size N_t (at birth) per generation. Time is discrete and indexed by $t = 0,1,2,....$ The length of each generation

is conventionally set at 20 years. An individual lives for three periods: childhood and adolescence, young adulthood (or adulthood, the working period) and old age. A new born of generation t may either die early i.e., before parents spend time for his education (this holds with probability $1-\Gamma_{t+1}$), or he may survive thus receiving education (e_{t+1}) according to parents' decisions. As an adolescent, still belonging to generation t, he does not make economic decisions and does not work, but can become sexually active and acquire HIV infection. Finally, if he survives, he becomes economically active at the beginning of adulthood (time $t+1$). The probability $\Gamma_{t+1} \leq 1$ that a newborn of generation t survives up to the onset of adulthood, $\Gamma_{t+1} \leq 1$, is assumed to positively depend on the level of the human capital of parents h_{t+1}, and to negatively depend on the prevalence of HIV i_{t+1}, i.e. $\Gamma_{t+1} = \Gamma(h_{t+1}, i_{t+1})$.

An adult may acquire HIV infection and takes relevant economic decisions. He has a probability $\Pi_{t+1} = \Pi(h_{t+1}, i_{t+1})$ to survive up to the onset of old age. This probability positively depends on the human capital of parents and negatively on HIV prevalence, i_{t+1}. Every adult agent is endowed with h_{t+1} units of human capital that are (inelastically) supplied to firms in exchange for wage income w_{t+1} per unit of labour. The (expected) lifetime utility function captures the individual preferences towards consumption (c_{t+1}) and the number of surviving children (n_{t+1}) during the first part of his economic life (adulthood), as well as the number and the quality of children during the second part of his economic life (old age) (see Fig. 2 for the timeline of events, which extends to our setting with endogenous fertility and endogenous mortality a similar picture from [Chakraborty et al. (2016)]). Finally, when entering old age, and individual lives period $t+2$ where he does not consume and gets utility just from his children (quantity) and their human capital endowment (quality).

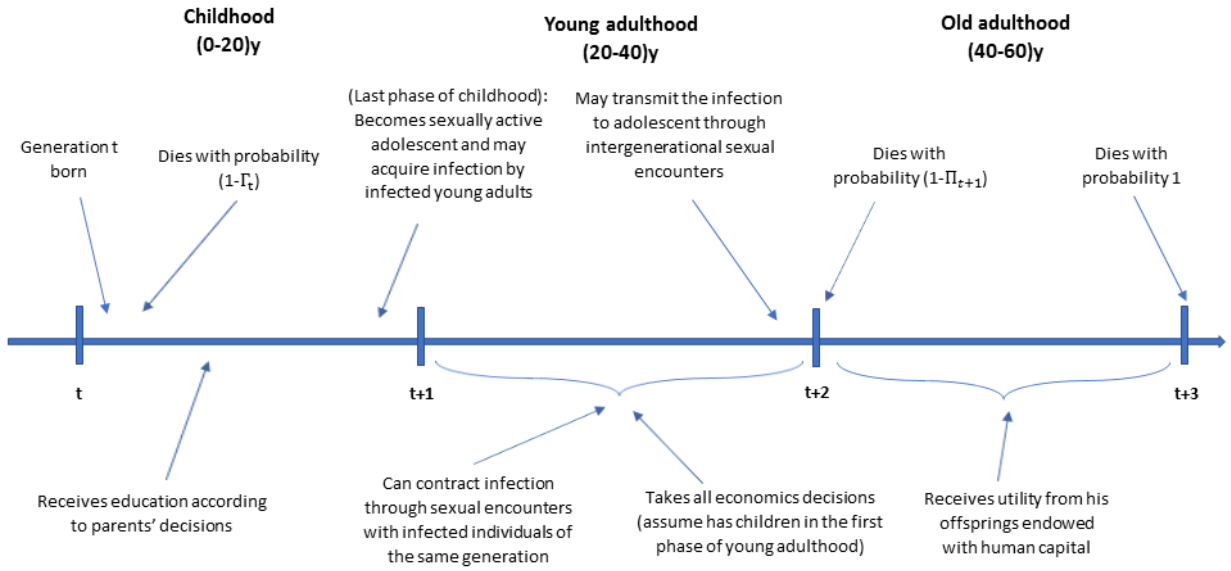

Fig. 2. Timeline of events. This figure summarises the sequence of economic and disease-related events for an individual born at time t who may become an economically and sexually active adult at time $t+1$.

3.2. Disease transmission

We represent HIV spread by following [Chakraborty et al. (2010, 2016)], who proposed a clever and parsimonious approach to represent a deadly epidemics as a persistent phenomenon, characterised by a specific temporal profile induced by the patterns of transmission between individuals, rather than a mere mortality shock with a predetermined duration, as instead is used in

the other contributions in the economic literature. In the subsequent model parametrization an effort has been made to harmonise the observed characteristic time scales of the fertility transition and the HIV/AIDS epidemics as observed in SSA, in order to represent both phenomena in a realistically correct relative time scale.

The adopted approach (Fig. 3) rests on the simplifying hypothesis that only infective adults contribute to retransmit the infection. In other words, HIV- susceptible individuals (be they in the adult or the adolescent phase) can acquire HIV infection through sexual contacts with HIV-infective adults only. This hypothesis avoids a main shortcoming that would follow (in OLG models) on the assumption that only adult individuals contribute to infection spread, namely the automatic extinction of the epidemics once adult individuals enter the old stage leaving no infective individuals to ensure the subsequent spread of the epidemics.

Let p_t be the probability that a young adult acquires HIV infection. This is a function of the prevalence in the population of generation t, that is

$$p_t = 1-(1-i_t\lambda)^{\mu}, \tag{1}$$

where $0<\lambda\leq 1$ is the constant probability of being infected per sexual partnership with an infected individual and $\mu>1$ represents the number of sexual partnerships during the adult period. If the population is sufficiently large, the prevalence rate at time $t+1$ amongst young adults converges to the probability of a young adult to be HIV-infected, i.e. $i_{t+1}=p_t$. Then,

$$i_{t+1} = 1-(1-\lambda i_t)^{\mu}. \tag{2}$$

Unlike [Chakraborty et al. (2016)], we do not link HIV spread to agents' behaviour. Indeed, we believed unreasonably strong assuming for SSA countries that individuals rationally choose health investments for HIV prevention, given their utility function and budget constraint. The ability to affecting such a mechanism is the main aspect to be considered for public policies (i.e., health investments provided by international organizations or private foundations, as the well-known Bill and Melinda Gates foundation) aiming to reduce the impact of the epidemics on the economic system.

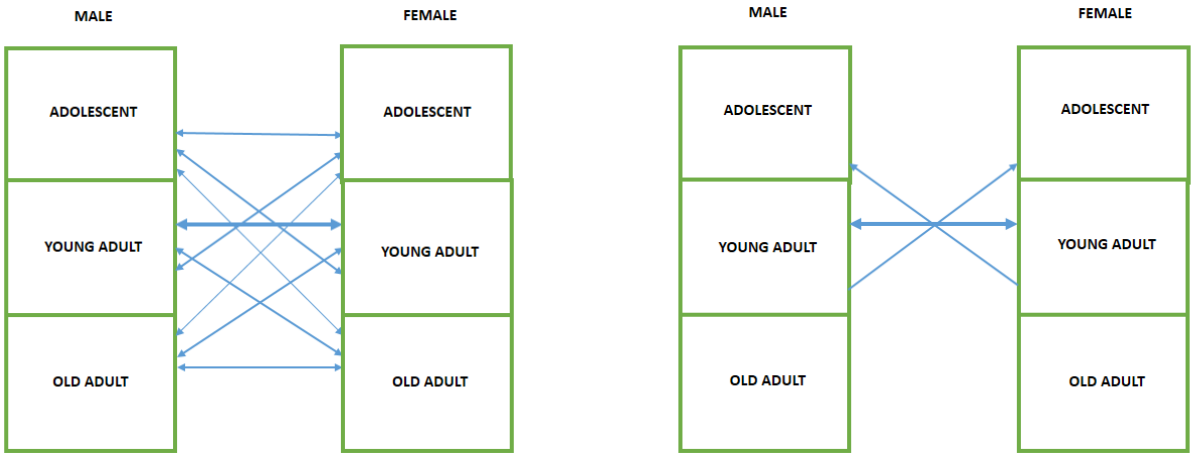

Fig. 3. Mechanism of diffusion of the infection. Left panel: realistic HIV transmission among age groups. Right panel: the stylised mechanism of diffusion considered in our model.

3.3. Preferences and solutions

By normalizing the utility from death to zero, preferences of the representative individual that is economically active at time $t+1$ are captured by an inter-temporal (expected) utility function whose formulation accords with the Beckerian tradition, that is:

$$U_{t+1} = \ln(c_{t+1}) + \rho\ln(n_{t+1}) + z\Pi_{t+1}\ln(n_{t+1}h_{t+2}), \tag{3}$$

where $\rho > 0$ captures the parent's relative taste for children and $z > 0$ is a scaling parameter tuning the relative degree of altruism. This allows receiving utility (with certainty) from material consumption and the quantity of children when young, whereas getting utility from both quantity and quality of children conditioned to the event of becoming old. This holds with probability Π_{t+1}. The quality of children is represented by their own level of human capital (education). Our decision of weighting the second flow of utility with the survival probability may have the traditional interpretation that in the case of premature death agents do not get any utility flow from their educated children. Another possible interpretation we can provide for this formulation of lifetime utility is the following: given the limitations imposed by the non-adoption of a model with heterogeneous individuals, this kind of preferences allows (on average) capturing the lower level of investment in education of orphans, which increases in number as a consequence of an increase in adult mortality. This is largely observed in high HIV prevalence rate SSA countries.

The relationship between the total number of born children and the number of surviving ones at every time $t \geq 0$ is given by:

$$n_t = \Gamma_t n_t^g \,. \tag{4}$$

A t-generation individual choice is made at time $t+1$ subject to the budget constraint:

$$c_{t+1} = w_{t+1}\left(m - \psi \frac{n_{t+1}}{\Gamma_{t+1}} - \phi_{t+1} n_{t+1} - e_{t+1} n_{t+1} \right), \tag{5}$$

where m is his time endowment. The budget constraint in (5) implies that consumption is constrained by the amount of resources available after accounting for the fraction of the time endowment for giving birth to n_{t+1}^g children ($\psi \in (0,m)$), and raising ($\phi_{t+1} \in (0,m)$) and educating (e_{t+1}) those who survive (n_{t+1}).

The human capital of each child depends on the human capital of parents and the time expenditure in education. Its evolution over time is driven by the following equation:

$$h_{t+2} = q(x + e_{t+1}) h_{t+1}^{\alpha}, \tag{6}$$

where $q > 0$ and $\alpha > 0$. Given that $x + e_{t+1} > 0$, it follows $h_{t+1} \in (0,+\infty)$ for every $t \geq 0$ as $h_1 > 0$ (the initial condition) must hold. The term $x > 0$ guarantees that children's human capital is positive even if parents do not invest in education.

The maximisation of the utility function with respect to material consumption, the number of children and time education investment, subject to the budget constraint, the accumulation of human capital and the condition $c_{t+1} > 0$, gives the solution to the optimisation programme. This solution may be either interior ($e_{t+1} > 0$) or on the corner ($e_{t+1} = 0$). The interior solution implies:

$$e_{t+1} = \frac{z\Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} - x \,, \tag{7}$$

$$n_{t+1} = \frac{m\rho\Gamma_{t+1}}{[\Gamma_{t+1}(\phi_{t+1} - x) + \psi](1 + \rho + z\Pi_{t+1})} \,, \tag{8}$$

$$c_{t+1} = \frac{m w_{t+1}}{1 + \rho + z\Pi_{t+1}} \,. \tag{9}$$

The condition $\Gamma_{t+1}(\phi_{t+1} - x) + \psi > 0$ must hold. In addition, the condition that guarantee the existence of an interior solution is $\frac{z\Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} > x$. Then, if $\frac{z\Pi_{t+1}[\Gamma_{t+1}(\phi_{t+1} - x) + \psi]}{\rho \Gamma_{t+1}} < x$, we get the corner solution, which is given by:

$$e_{t+1} = 0 \,, \tag{10}$$

$$n_{t+1} = \frac{m\Gamma_{t+1}(\rho + z\Pi_{t+1})}{(\phi_{t+1}\Gamma_{t+1} + \psi)(1+\rho+z\Pi_{t+1})}, \tag{11}$$

$$c_{t+1} = \frac{mw_{t+1}}{1+\rho+z\Pi_{t+1}}. \tag{12}$$

All the choices depend on the parameters as expected. What is important to stress is the dependence of the optimal values of education and fertility on the adult survival probability. An increase in this probability reduces fertility and raises the time investment in education per child. The reason for the switch in the allocation of resources from quantity to quality of children depends on the higher probability to receiving a utility inflow in old age. The main effect for the next period output is a higher level of human capital due to a larger investment in education when individuals live longer. This mechanism allows capturing the main stylised facts as well as the main dynamics of the demographic transition observed in the empirical evidence including the quantity-quality trade-off, which represent one of the main causes for economic development.

The production of final output (Y_t) in the economy occurs under perfect competition according to the technology $Y_{t+1} = H_{t+1} + \theta L_{t+1}$, where $H_{t+1} = h_{t+1}L_{t+1}$ is the aggregate stock of human capital at time *t+1*, L_{t+1} is the labour input in the same period, and $\theta \geq 0$ is a parameter capturing the worker's productivity. By normalising to 1 the price of Y_{t+1}, profit maximisation implies that labour is paid according to its marginal product, that is $w_{t+1} = \theta + h_{t+1}$.

By using the interior or corner solution together with the equation describing human capital accumulation and the expression of the wage rate, we obtain two-dimensional map describing the evolution of human capital and HIV prevalence over two subsequent periods, which completely characterise the dynamics of the economy, i.e.:

$$h_{t+1} = \begin{cases} qxh_t^{\alpha} \quad if \quad e_t = 0 \\ \dfrac{qz\Pi_t[(\phi_t - x)\Gamma_t + \psi]}{\rho\Gamma_t} h_t^{\alpha} \quad if \quad e_t > 0 \end{cases}. \tag{13}$$

$$i_{t+1} = 1 - (1 - \lambda i_t)^{\mu}$$

3.4. Intervention programs

Intervention programs against HIV are modelled in the simplest manner i.e., by a fully exogenous reduction in the transmission probability per partnership λ. This is assumed for simplicity to be funded by external interventions (e.g., by international organizations), as has often been the case [WHO 2016]. This can capture a range of different medical (e.g., anti-retroviral treatment having the potential to reduce infectivity) as well as non-medical interventions (e.g., aimed to increase awareness of risk and favour spontaneous behaviour change, such as reducing the number of sexual partners and increasing use of condoms). The intervention is assumed to be introduced at time $t = T$ as follows:

$$\lambda_t = \begin{cases} \lambda \quad if \quad t < T \\ \lambda + (\lambda_l - \lambda)\dfrac{f(t-T)}{1+f(t-T)} \quad if \quad t \geq T \end{cases}. \tag{14}$$

Where f is a parameter capturing the effectiveness of the policy over time, and λ_l summarises the strength of the policy in terms of reduction in the transmission probability.

3.5. Model parametrisation

3.5.1. Functional forms for child and adult survival

To cope with a context of demographic transition with endogenous mortality and HIV spreading, we let child and adult survival probabilities depend on both human capital and HIV prevalence. In particular, we model the child survival probability Γ_{t+1} as follows:

$$\Gamma_{t+1} = \xi_{t+1}(1-\delta i_{t+1}), \tag{15}$$

where ξ_{t+1} represents the child survival probability in the absence of HIV and $0<\delta\leq 1$ is the conditional probability that a child of HIV-infected parents is vertically transmitted at birth (and of dying soon after), so that δi_{t+1} is the unconditioned probability for a child to be born infected (and of dying soon after). In addition, we adopt the following functional for ξ_{t+1}:

$$\xi_{t+1} = \Gamma_{pre} + \left(\Gamma_{post} - \Gamma_{pre}\right)\frac{b\ln(1+h_{t+1})}{1+b\ln(1+h_{t+1})}, \tag{16}$$

where $\Gamma_{pre}, \Gamma_{post} \in [0,1]$ are the child survival at birth in the Malthusian regime prevailing before mortality decline and the child survival at birth in the modern regime at the end of the mortality transition [Galor (2005, 2011)], and $b>0$ is a tuning parameter which governs the shape of the mortality transition pattern along the growth of human capital. The functional form (16) depicts a pattern of increasing child survival during the mortality transition, departing from its Malthusian level Γ_{pre} and eventually ending at its modern level Γ_{post} by the end of the transition. The temporal pattern of the (endogenous) mortality transition is modelled through a logarithmic function of human capital, but many other functions could play the same role.

Similarly, the probability Π_{t+1} to reach old age conditioned on being entered the adult state is modelled as follows:

$$\Pi_{t+1} = \left[\Pi_{pre} + \left(\Pi_{post} - \Pi_{pre}\right)\frac{B\ln(1+h_{t+1})}{1+B\ln(1+h_{t+1})}\right](1-i_{t+1}), \tag{17}$$

where $\Pi_{pre}, \Pi_{post} \in [0,1]$ and $B>0$ have the corresponding meaning of Γ_{pre}, Γ_{post} and b in Eq. (16). Finally, the cost of raising children is represented as an increasing function of human capital accumulation and its form resembles those adopted in (16) and (17).

$$\phi_{t+1} = \phi_{pre} + \left(\phi_{post} - \phi_{pre}\right)\frac{\ln(1+h_{t+1})}{1+\ln(1+h_{t+1})}. \tag{18}$$

3.5.2. Parameter assignment

Summary information about model parameters is reported in Tab. 1. Demographic parameters were drawn from the post-1950 demographic experience of SSA countries [UN (2015)]. The HIV equation was parametrised by assigning the involved parameters (μ, λ) in order to reproduce at equilibrium the range of prevalence observed in medium-high prevalence SSA countries according to the last UNAIDS estimates of HIV prevalence in the adult population [UNAIDS (2016)]. In most case, UNAIDS estimates show a constant trend for several years before 2015 (Fig. 1a). This suggests the achievement of an endemic quasi-equilibrium, which supports our strategy. Economic parameters were either borrowed from the literature or assigned as simulation parameters.

Parameter	Description	Value or range	Source
μ	Average number of sexual partners per adult individual during the entire adult stage	450-500	Assigned to match predicted equilibrium prevalence and UNAIDS estimates.
λ	Conditional transmission probability of HIV per single adequate sexual contact	[0.0022,0.0028]	Assigned to match predicted equilibrium prevalence and UNAIDS estimates.
δ	Probability that a newborn is vertically transmitted from HIV-positive parents.	0-0.25	[Chakraborty et al. (2010, 2016); WHO (2016)]
z	Relative degree of individual's altruism	70	Assigned to match pre-transition level of

			TFR [UN (2015)]
ρ	Parent's taste for children	2.04	Assigned to match pre-transition level of TFR [UN (2015)]
m	Time endowment of households	5	Simulation
θ	Coefficient capturing natural endowment of rural economy	8	Simulation
ψ	Time cost of bearing a child	0.002	Simulation
ϕ_{pre}	Parameter regulating pre-transition time cost of rearing a child	0.394	[30,31]
ϕ_{post}	Post-transition time cost of rearing a child	0.475	[Haveman and Wolfe (1995); de la Croix and Doepke (2004)]
v	Speed of change of the time cost of rearing	1/8	Simulation
Γ_{pre}	Parameter tuning pre-transition child survival probability	0.1381	[UN (2015)]
Γ_{post}	Post-transition child survival probability	0.9	[UN (2015)]
b	Speed of child mortality transition	1	Simulation
Π_{pre}	Parameter tuning pre-transition adult survival probability	0.5767	[UN (2015)]
Π_{post}	Post-transition adult survival probability	1	[UN (2015)]
B	Speed of adult mortality transition	1	Simulation
α	Relative weight of parental human capital in the production of children's human capital	1	[de la Croix and Doepke (2004)]
q	Coefficient of the human capital technology	0.6	[de la Croix and Doepke (2004)]
x	Level of automatic transmission of human capital	0.34	Simulation
f	Effectiveness of the policy over time	0.01	Simulation
λ_1	Strength of the policy in reducing the transmission probability	0.0001	Simulation

Tab. 1. Summary information about model parameters; t denotes time in OLG units i.e., it represents the length of an OLG generation (set to 20 years in the model).

4. Results

4.1. The case of a large, uncontrolled, HIV epidemics

Under the adopted parametrisation, the model predicts – in the absence of HIV – the onset and gradual completion of the fertility transition as an endogenous response to mortality decline through the increase in education investments, with the TFR declining from about six down to replacement level (about 2.1), in line with UN projections medium variant (Fig. 4, left axes).

As for the HIV epidemics, we first considered the following idealised scenarios without any control interventions, where the infection gradually increases up to an endemic equilibrium prevalence (EP) and persists there: "low": EP=10%, "medium": EP=15%, "high": EP=20%, "very high": EP=30%), (Fig. 4, right axes). These scenarios bound UNAIDS estimates [UNAIDS (2016); WHO (2016)] for countries with large epidemics. In particular, Fig. 4 aims to reflect a potentially important factor, namely the variability in the stage of the fertility transition by SSA countries at the onset of HIV epidemics, namely the case where HIV took-off at an advanced stage of the fertility transition (Fig. 4, left panel), as occurred for South Africa, vs the case where HIV onset occurred at an earlier stage (Fig. 4, right panel).

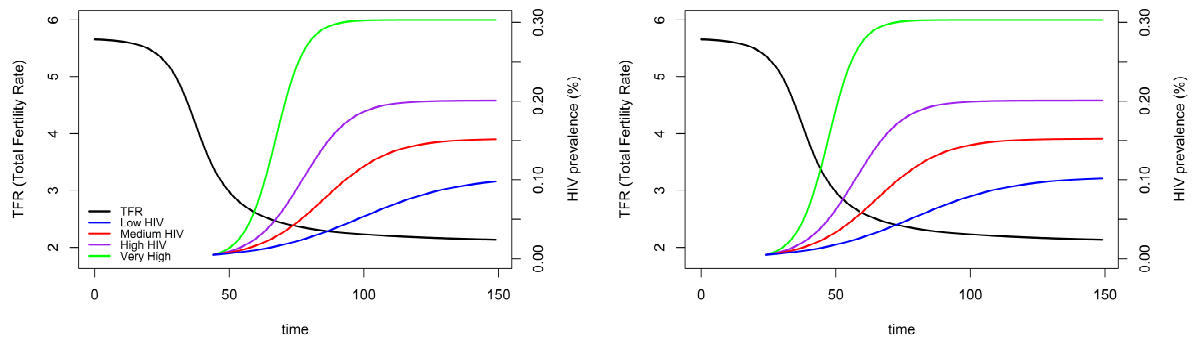

Fig. 4. The hypothesised trajectories of SSA fertility in the absence of HIV (left axis) and of HIV prevalence (right axis) under uncontrolled HIV epidemics of different intensity. Left panel: the onset of HIV occurs at an advanced stage of the fertility transition; right panel: the onset of HIV occurs at an earlier stage of the fertility transition.

The predicted impact of a persistent uncontrolled HIV epidemic on the fertility transition in SSA is dramatic (Fig. 5), with fertility landing on levels well above the replacement threshold of 2.1. For high levels of the EP, the TFR eventually stalls on levels in excess of three. Moreover, medium-high HIV epidemics have the potential to cause a reversal in the fertility transition pathway. Two main qualitative scenarios emerge, depending on whether the onset of HIV occurred either at an advanced or at an earlier stage of the fertility transition (Fig. 4). In the former case, at the onset of HIV the TFR has already experienced a substantial decline because much of the gain in survival was already achieved. The reversal is predicted to be sharp, with a marked increase in TFR that remains essentially constant thereafter. In the latter case, the TFR shows a temporary relapse, after which it resets on a declining path. The difference is because in the latter case fertility decline triggered by mortality progress from causes of deaths different from HIV was just initiated, so that substantial room for further fertility decline actually remained.

The causation chain (illustrated in Fig. 6 for the scenario of late onset of HIV) goes as follows: (a) HIV breaks-down the long-term declining trend in adult mortality, which suddenly upturns (Fig. 6a); the upturn in adult mortality reverses the positive trend on education investments (Fig. 6b); (c) the collapse in education investment sets the growth rates of human capital at levels, which are insufficient to further fuel the quantity-quality switch, and therefore to further promote fertility decline (Fig. 6c).

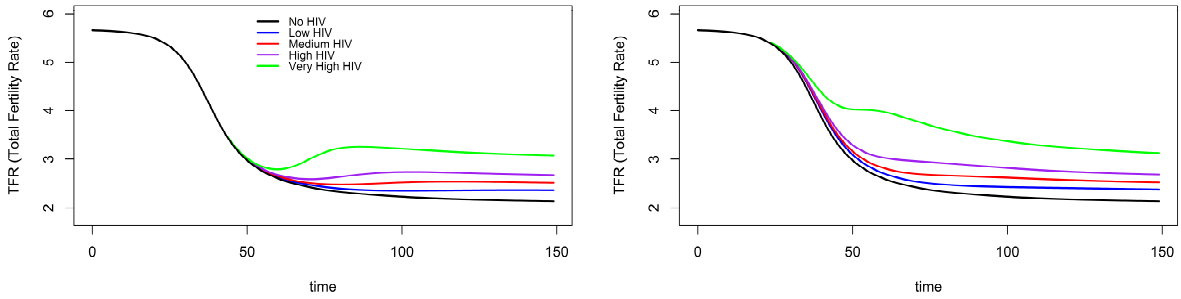

Fig. 5. Potential impact of uncontrolled HIV epidemics on the fertility transition in SSA for different levels of HIV prevalence. Left panel: HIV onset occurs at an advanced stage of the transition; right panel: HIV onset occurs at an earlier stage of the transition.

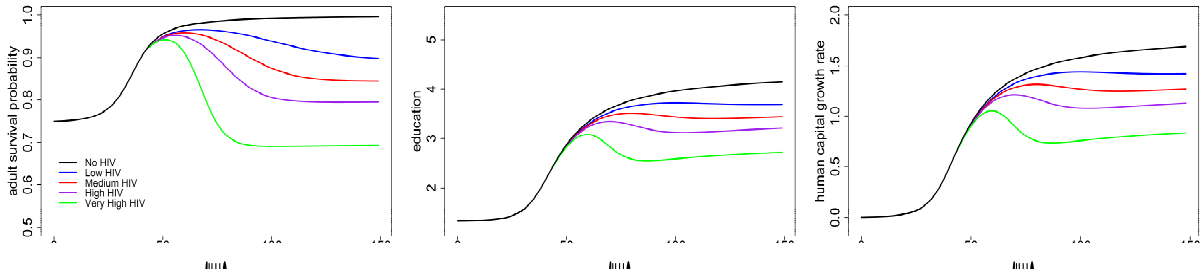

Fig. 6. The causation chain in the predicted fertility reversal in SSA under an uncontrolled HIV epidemics. The figure reports the temporal trends of the adult survival probability (left), of the education index (centre), and of the growth rate of human capital (right).

4.2. The effects of interventions

The previous analysis described a useful theoretical benchmark, against which grounding the possible effects on fertility of the current realistic scenarios of HIV control [WHO (2013)]. For the sake of simplicity, we only consider here fully effective interventions, that is interventions capable to eventually eliminate HIV, but characterised by different timings. More precisely, we compare an early intervention, initiating shortly before the epidemics has reached its equilibrium prevalence, with a number of delayed interventions (Fig. 7). In particular, we hypothesise that the time span needed to bringing full HIV to elimination from the intervention onset is coarsely comparable with the time span that the HIV epidemics took to reach its peak (Fig. 7, left panel), which in SSA lasted approximately 2-3 decades [UNAIDS (2016)]. This hypothesis is motivated by the fact that in SSA a large proportion of currently seropositive individuals is still unaware of his health status and might therefore remain infective for decades [WHO (2016)].

Predictions indicate that bringing HIV mortality under full control will remove obstacles to investment in education so that fertility will eventually land to its replacement level (Fig. 7, right panel). Nonetheless, even under the early interventions scenario, the time span needed for bringing HIV under control, has the potential to cause a serious delay in the completion of the fertility transition. Obviously, this scenario can substantially worsen in the presence of delays in the effectiveness of interventions (Fig. 7, right panel).

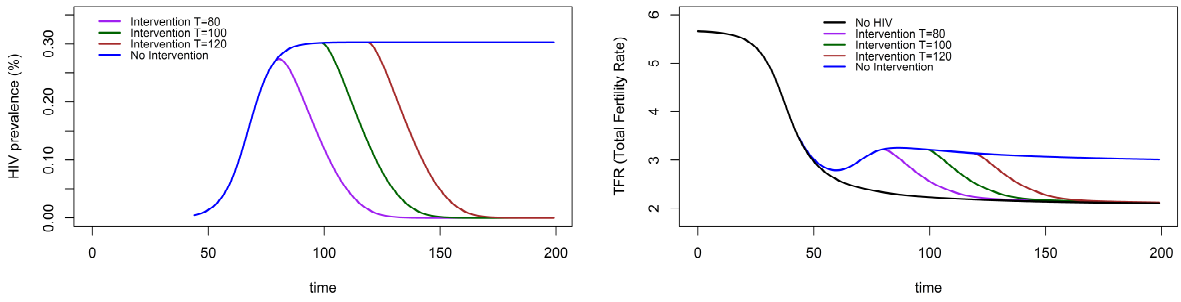

Fig. 7. The case of HIV onset at an advanced stage of the fertility transition. Temporal trend of HIV prevalence (left panel) and total fertility (right panel) under different effective intervention programs capable to eventually achieve HIV elimination. The intervention programs considered differ in the timing of initiation (t=80,100,120).

4.3. Substantive implications and further discussions

Despite its poorest health conditions, with the highest impact of communicable diseases worldwide [Bloom and Canning (2004)], SSA experienced continued – though slow – mortality decline until the onset of HIV/AIDS. In high-prevalence countries HIV has become the leading cause of mortality [IHME (2013)], capable to reversing the increasing trend in life expectancy. However, the ultimate effects of HIV on fertility and economic development in SSA are more controversial, as

discussed in Section 2. This work has built on a simple macroeconomic framework including HIV spread, endogenous child and adult mortality, endogenous fertility and private educational investment, to be used (i) as a baseline to reassess the ultimate impact of HIV on fertility in SSA, in the light of the recent advances on the economic determinants of fertility transitions [Galor (2011)], (ii) as a support to prioritise future economic intervention programs against HIV based on a more general societal perspective.

The adopted parametrisation is robust on the timing of the interplay between HIV and the fertility transition in that it keeps in a correct empirical balance the relative time scales of the epidemic and of the fertility transition. In the absence of HIV, the model predicts the completion of the fertility transition as an endogenous response to the decline in both young and adult mortality, which in turn promotes investments in education and eventually fertility decline via the quantity-quality switch.

In the theoretical case of a large uncontrolled HIV epidemic (say, yielding an endemic prevalence above 15%), HIV has full potential to prevent the completion of the fertility transition. In particular, reversals in the fertility trajectory are more likely to occur in countries where the onset of HIV occurred at a later stage of the fertility transition. However, the ultimate level of fertility i.e., how far the TFR will eventually land from the replacement level, only depends on the intensity of the HIV epidemic and not on the stage of the transition where the epidemics actually debuted. The critical obstacle to the completion of the fertility transition is the AIDS-related increase in adult mortality, which reduces the parents' educational investment on children and therefore prevents the quantity-quality switch.

Hopefully, the increasing adoption and effectiveness of intervention against HIV/AIDS worldwide [UNAIDS (2016), WHO (2016)] should make an uncontrolled HIV epidemic only a theoretical worse case. Our results however show that, though achievement of HIV full control would remove the obstacles to the eventual completion of the fertility transition, the timing with which HIV control measures will be enacted becomes critical. Indeed, any delay in controlling HIV would not only allow the direct negative effects of HIV, namely its large mortality burden, to persist, but might also cause fertility to remain persistently high due to the decline of private investment in education. In addition, the persistence of high fertility would promote continued population growth, therefore loosing epochs potentially favourable to economic development.

We believe that the relationships between HIV, education and fertility theoretically inferred here should be carefully considered on a policy standpoint. This is because 1) the recent empirical evidence shows a robust negative relationship between HIV prevalence and education [Fortson (2011); Akbulut-Yuksel and Turan (2013)]; 2) the time needed for full HIV control depends of the effectiveness and diffusion of interventions, which in turn depend on the resources allocated for HIV control. Given the huge number of seropositive individuals in SSA [UNAIDS (2016); WHO (2016)], most of which unaware of their sero-status, this is hard to predict for SSA. From this standpoint there seems to be only one possible policy recommendation for international interventions: the maximal control effort on HIV should be done right now, without delays, and with priority to those situations e.g., high-prevalence countries as Lesotho and Swaziland, where the long-lasting mortality crisis due to HIV, which is projected to require several decades before being reabsorbed, could threaten the fertility transition. This also makes it important emphasizing the danger of conservative approaches, as in the last round of the UN projections, where the possible feedbacks of HIV on fertility in SSA were deliberately ruled out [UN (2015)].

Further extensions of the present work should consider model-based frameworks including 1) saving and accumulation of physical capital (deliberately avoided here to mirror SSA economies, which show the lowest aggregate saving [World Bank (2016)], the highest income inequality [IMF (2015)], and the most under-developed political and financial systems worldwide [Easterly and Levine (1997)]); 2) continuous-time to realistically capture the time scales of the interplay between HIV and fertility; 3) the impact of the HIV/AIDS on labour productivity, as in Chakraborty (2010, 2016).

5. Conclusions

This article tackled one of the most important open questions on economic development, namely the impact of HIV/AIDS on the fertility transition in Sub-Saharan Africa. All successful stories of economic development initiated by mortality decline, which in turn triggered the decline of fertility, thus making resources available for investments in education and fuelling a virtuous circle of sustained development [Becker et al. (1990); Fogel (2004); Galor (2005, 2011); Livi-Bacci (2017)]. In some Sub-Saharan African (SSA) countries suffering large HIV/AIDS epidemics, life expectancies are projected to return to pre-HIV levels only by 2060 [UN (2015)], fertility decline is stalling [UN (2015)], and there is evidence of negative effects of such a disease on educational investments [Fortson (2011); Kalemli-Ozcan (2012)]. This research built upon these facts by reporting model-based evidence that even under successful disease control in the long term, HIV might substantially delay the fertility transition in SSA by halting economic development for decades. The results challenge the optimistic view, which seems to be adopted amongst international institutions due to the increasing degree of epidemic control. In particular, our research predicted that in SSA HIV/AIDS still has a large room for compromising the children quantity-quality switch, which has represented the major trigger of economic development all over the world. This possibility should receive the highest priority among international institutions.

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